TETANUS IN A PARENTERAL DRUG ABUSER: REPORT OF A CASE

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Tetanus is an infection caused by *Clostridium tetani.* In the United States, tetanus remains a significant problem primarily among nonimmunized or inadequately immunized individuals.

This article reports a fatal case of tetanus that occurred in a 45-year-old parenteral drug abuser who presented to Harlem Hospital Center with nuchal rigidity, trismus, dysphagia, and spasms of the pectoralis musculature. Multiple cutaneous ulcerations also were observed. Despite aggressive measures that included: endotracheal intubation, administration of human tetanus, hyperimmune globulin, tetanus toxoid, and intravenous penicillin, the patient rapidly deteriorated and manifestations of heightened sympathetic nervous system activity, seizures, and cardiac arrest ensued.

The diagnosis of tetanus must be based upon clinical grounds. Clinicians must remain aware of the possibility of tetanus, especially among drug abusers who also are most likely to be evaluated for complications of human immunodeficiency viral infection, which in some cases may mimic tetanus or make the diagnosis more difficult to establish. (*J Natl Med Assoc.* 1994;86:223-225.)

Key words • tetanus • parenteral drug abuse

The clinical manifestations of tetanus are caused by tetanospasmin, an extremely potent exotoxin, which is elaborated by *Clostridium tetani.* This disease has affected humans for thousands of years. Hippocrates describes a case in which he initially uses the term opisthotonos.[1] Here in the United States, tetanus remains problematic among individuals who are nonimmunized or incompletely immunized. The incidence of tetanus has not changed significantly during the last 15 years, following a steady decline between 1947 and 1976. In all likelihood, this trend could be attributed to improved wound management and national immunization programs.[2]

According to the Centers for Disease Control (CDC), 101 cases of tetanus were reported between 1987 and 1988, with an annual incidence of 0.02/100 000. Of these 101 cases, six occurred in parenteral drug abusers.[3]

CASE REPORT

A 45-year-old man with a history of cocaine (crack) abuse and parenteral and subcutaneous (skin popping) heroin abuse presented to Harlem Hospital Center with nuchal rigidity, trismus, dysphagia, and epigastric pain of 24 hours' duration. Vital signs were temperature 97.3°F, respiratory rate 18/min, blood pressure 130/90 mm Hg, and pulse 80/min.

Physical examination revealed marked nuchal rigidity, spasms of the upper extremities, and pectoralis musculature. There were multiple ulcerated areas over the trunk and extremities. The deep tendon reflexes were hyperactive.

Clinical laboratory values were within normal limits. The cryptococcal antigen was negative and blood and cerebrospinal fluid cultures were negative. The CD4/CD8 cell counts were 911/mm^3 over 611/mm^3 = 1.4.

The diagnosis of generalized tetanus was suspected on the basis of clinical findings. The patient was treated with penicillin G intravenously, tetanus toxoid 0.5 mL intramuscularly, and human tetanus hyperimmune globulin 3000 units. A tracheostomy was performed on

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TABLE 1. DIFFERENTIAL DIAGNOSIS OF TETANUS

Disease	Distinction of Tetanus
Meningitis	Headache, nuchal rigidity, photophobia, Kernig's and Brudzinski's signs, abnormal cerebrospinal fluid analysis
Strychnine poisoning	Test urine for the presence of strychnine
Phenothiazine drug toxicity	Response to anticholinergic agent such as benztropine
Drug withdrawal (opiates)	Impaired mental status, dilated pupils, piloerection
Status epilepticus	Tonic-clonic activities, electroencephalogram results
Hypocalcemic tetanus	Decreased calcium levels, Trousseau's and Chvostek's signs
Peritonsillar abscess	Physical findings, dental radiographs
Subarachnoid hemorrhage	Computed tomography scan and cerebrospinal fluid analysis (if CT scan results are nondiagnostic)

the third hospital day and the patient was given pancuronium intermittently to control spasm. The hospital course was complicated by *Acinetobacter* pneumonitis, pulmonary aspiration, and multiple urinary tract infections. The patient remained febrile despite broad spectrum antibiotic coverage for over 2 weeks during the hospital stay. He was treated empirically with amphotericin B with a good clinical response. He sustained a cardiac arrest on the 13th hospital day, and he was successfully resuscitated. Subsequent therapy included propanolol and clonidine for hypertension, mechanical ventilation, and total parenteral hyperalimentation. The patient, however, sustained a second cardiac arrest and expired during the 13th week of hospitalization.

DISCUSSION

It is well known that parenteral drug abusers are prone to acquired immunodeficiency syndrome (AIDS), endocarditis, viral hepatitis, septicemia, and malaria, but there are few reports in the literature dealing with tetanus in this population. Cherubin[4] reported one of the largest series of tetanus in the heroin addicts. He indicated a greater preponderance of women with tetanus in New York City. He speculated that women may have less effective immunization and that subcutaneous injections (skin popping) complicated by chronic ulcerations put these women at a greater risk. Of note, the first case of tetanus in a morphine addict was reported in literature over a century ago.

Tetanus is a potentially fatal infection caused by the exotoxin tetanospasmin, which is produced by *C tetani.* This organism is a gram-positive, anaerobic, spore-forming bacillus, which is ubiquitous: it is present in soil and dust and often is found within the intestinal tracts of animals. Tetanus may develop when lacerations, fractures, burns, hypodermic injection sites, or umbilical stumps become contaminated with bacterium. With subcutaneous injections of heroin, which often contain quinine, anaerobic conditions are enhanced leading to optimum conditions for the growth of *C tetani.*[5] As a result, a neurotoxin is produced, which may affect neurotransmitter release by binding the presynaptic inhibitory synapses, resulting in increased muscle tone and rigidity.

The diagnosis of tetanus is based upon clinical grounds. Cultures are nearly always negative. Brust and Richter[6] reported that from 30 heroin addicts with tetanus at Harlem Hospital, none of the wounds showed *C tetani* on smear or grew on anaerobic cultures. They concluded that the combination of multiple wounds, competing organisms, and delays between debridement and anaerobic culturing probably accounted for their zero yield. Hence, it is crucial to recognize the clinical manifestations of tetanus because of its high mortality.

The majority of patients will present with trismus and paraspinal rigidity. Other manifestations include: dysphagia, neck stiffness, spasms of the face, trunk, and chest wall, a facial grimace (risus sardonicus) due to persistent trismus, opisthotonos due to persistent spasms of the back musculature, flexion and adduction of the arms clenching on the thorax, extension of the legs, lethargy, irritability, and restlessness. They also may present with laryngeal and glottal spasms causing cyanosis and asphyxia. The diaphoresis and muscle exertion that accompany these seizure-like movements lead to fever.[7]

The differential diagnosis of tetanus includes: meningitis, strychnine poisoning, status epilepticus, hypocalamic tetany, phenothiazine drug toxicity, drug withdrawal (opiates), peritonsillar abscess, and subarachnoid hemorrhage (Table 1). Clinicians must be alert to the possibility of tetanus in parenteral drug users where the complications of human immunodeficiency virus infection may make the diagnosis more difficult to establish.

TREATMENT

In the United States, tetanus is a preventable disease

in most cases. Vaccination with a primary series of three doses of tetanus toxoid and booster doses every 10 years is highly effective in the prevention of tetanus. Recommendations for prophylaxis in wound management are given in Table 2.[2,3] Failures of tetanus toxoid can be due to: agammaglobulinemia, immunosuppressive drugs, carcinoma of the breast, poor toxoid, and inaccurate toxoid history. Individuals with AIDS may not respond to the initial injection of tetanus toxoid.[8]

Once the diagnosis of tetanus is established, therapeutic protocols concerning immunotherapy, antibiotic treatment, wound debridement, muscle relaxation, and respiratory and cardiovascular management should be addressed. The patient should be placed in a quiet environment because many types of stimuli may worsen the convulsive spasms. Human tetanus immunoglobulin should be given in order to neutralize the circulating toxin before it reaches the nervous system. Tetanus toxoid to stimulate effective active immunizations should be given as well. Penicillin is the recommended antibiotic, but other effective antibiotics are: metronidazole, tetracycline, erythromycin, and clindamycin. It should be emphasized that the course of the disease does not appear to be affected with antibiotic treatment because the toxin is already bound to the tissues. Extensive wound debridement should be done as soon as the antibiotic is administered. Abscesses should be irrigated with hydrogen peroxide. Sedatives like phenobarbital and chlorpropazine should be given to control spasms and decrease muscle rigidity. Diazepam may be added if spasms persist in some patients, while others require a neuromuscular blockade like pancuronium. Endotracheal intubation is frequently needed for airway control. Curare may be used for severe cases in low doses to minimize hypotension.[9]

IMPLICATIONS AND CAUSES OF DEATH

All patients with tetanus need supportive care in an intensive care unit setting because they require optimal nursing care. They are prone to have aspiration pneumonitis, hypoxemia, gastrointestinal bleeding, tracheal ulceration, sepsis, myopathy, pulmonary embolism, thrombophlebitis, renal failure, congestive heart failure, and extreme ranges of blood pressure between hypotension and hypertension. These conditions should be managed accordingly. Metabolic abnormalities are frequent and central hyperalimentation should be given.

Furthermore, respiratory failure, heart block with bradycardia or tachycardia and especially asystole, and ventricular fibrillation are common causes of death in some series, while asphyxiation secondary to spasms are common in others.

TABLE 2. TETANUS PROPHYLAXIS IN ROUTINE WOUND MANAGEMENT*

History of Absorbed Tetanus Toxoid	Clean Minor Wounds		All Other Wounds	
	Td	TIG	Td	TIG
Unknown or <3 doses	Yes	No	Yes	Yes
>3 doses	No	No	No	No

Abbreviations: Td = tetanus–diphtheria (adult Td) and TIG = tetanus immunoglobulin.
*From references 2 and 3.

Tracheostomy should be performed routinely in almost all the heroin addicts with moderate to severe cases of tetanus because of the occurrence of convulsive spasms, cardiorespiratory dysrhythmia, and hyperthermia.[8] Other indications for a tracheostomy are pulmonary atelectasis and inability to clear oropharyngeal secretions.[9]

CONCLUSION

Tetanus, a preventable disease, remains a challenge to the physician because the diagnosis is made on clinical grounds. It is critical that the signs and symptoms are well known because immediate measures need to be taken to decrease morbidity and mortality. This is one of many diseases likely to be encountered in parenteral drug abusers that physicians should be aware of it.

Acknowledgments

The authors thank Drs J. Austin, G. Comer, B. McCoy, and C. Francis for their comments.

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